THE VALSALVA MANŒUVRE IN AORTIC VALVE DISEASE

BY

A. E. DOYLE* AND G. H. NEILSON†

From the Department of Medicine, Postgraduate Medical School, Hammersmith

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Since the introduction of aortic valvotomy, the assessment of aortic valve disease has become increasingly important. Studies of arterial pressure pulse tracings, although useful in diagnosis (Goldberg *et al.*, 1954) have proved disappointing in assessing the severity of stenosis (Gorlin and Case, 1956). It is noticeable that the systolic upstroke time varies in patients with atrial fibrillation, the variation being apparently related to change in pulse pressure (Goldberg *et al.*, 1954).

This communication reports the effects of the Valsalva manœuvre on the arterial pressure pulse curves of forty patients with aortic valve disease, with particular reference to the relationship between the variations in systolic upstroke time and pulse pressure.

MATERIAL AND METHODS

Femoral artery punctures were performed with local anaesthesia in forty patients using a No. 18 Courmand needle, connected by a short length of polythene tubing (bore 2.0 mm.) to a Statham 606 strain gauge. Pressure pulses were amplified by a two-stage D.C. amplifier, and recorded on an Elmqvist Triplex recording galvanometer. The frequency response of this system has already been reported (Bayliss *et al.*, 1950).

The needle was directed centrally and held in this position by the operator. Failure to do this resulted in tracing artefacts. The patient was asked to blow into a pressure bottle; the pressure achieved was measured by a mercury manometer. Straining was continued for at least ten seconds.

Pulse pressures, and the time from the beginning of the rise of the pressure pulse curve to systolic peak, were measured before and during the period of straining. Pressure pulses recorded during the overshoot (Sharpey-Shafer, 1953) were discarded; as will be discussed later, the relationship between systolic upstroke time and pulse pressure altered during the overshoot. Variations in pulse form occasionally led to difficulty in measurement of these factors.

The femoral pulse was recorded in forty patients. Twenty of these had aortic stenosis, either alone or with insignificant aortic incompetence. Ten others had aortic stenosis and incompetence; and the remaining ten had aortic incompetence alone.

All the patients with aortic stenosis had typical systolic ejection murmurs (Leatham, 1951). All had some clinical, radiological, or cardiographic evidence of left ventricular hypertrophy. None had systolic blood pressures greater than 150 mm. Hg. Two had faint aortic diastolic murmurs. Thirteen were regarded as severe: all these had considerable exertional dyspnoea, ten had angina of effort, and the cardiograms showed marked T wave inversion in twelve (Abdin, personal communication). Three died and autopsy revealed severe aortic stenosis in each; the other ten had aortic valvotomy, and systolic gradients across the aortic valve ranged between

* Present address: Department of Medicine, University of Melbourne, Victoria.

† Travelling Scholar, Royal Australasian College of Physicians. Present address: Department of Medicine, University of Queensland.

30 and 150 mm. Hg. The absence of simultaneous estimation of left ventricular stroke volume makes these measurements difficult to evaluate, as is obvious from the tracings obtained at operation shown in Fig. 1, in which the gradient varies between 5 and 60 mm. from beat to beat in a case

FIG. 1.—Variations of the gradient across the aortic valve in a patient with severe aortic stenosis and atrial fibrillation. The variations correspond with those in pulse pressure. Time marker interval 0.04 sec. Note also the variations in upstroke time in the aortic pulse.

with atrial fibrillation. Of the remaining seven, who were regarded as having mild or moderate aortic stenosis, two had angina of effort and dyspnoea on exertion, and one angina alone. Cardio-graphic changes were less in this group. In the severe group the resting systolic upstroke time ranged from 0.17 to 0.27 sec., and in the milder group from 0.14 to 0.22 sec. In the severe group the pulse pressure ranged from 35 to 70 mm., and in the milder group from 46 to 92 mm.

Ten other patients with the signs of aortic stenosis had loud aortic diastolic murmurs. In this group the resting systolic upstroke times ranged from 0.08 to 0.22 sec., and resting pulse pressures from 42 to 102 mm. In the remaining ten who had pure aortic incompetence, the resting systolic upstroke times ranged from 0.06 to 0.10 sec., and the resting pulse pressures from 41 to 117 mm.

RESULTS

The Resting Pulse Tracing in Aortic Valve Disease. The distribution of resting systolic upstroke times and pulse pressures in each group is shown in Fig. 2 and the relationship between the two measurements is shown in Fig. 3. Although patients with aortic stenosis tend to have low resting pulse pressures, and although those with severe stenosis more commonly have lower pulse pressures than those with milder stenosis or associated incompetence, there is nevertheless a considerable overlap between the groups. The systolic upstroke time seems to be constantly prolonged in severe stenosis, but is often prolonged to a comparable degree in less severe cases or in combined lesions also. By contrast the systolic upstroke time is constantly short in those with pure aortic incompetence; in this group also, however, the pulse pressure varies widely.

Changes in Pulse Form during and following the Valsalva Manœuvre. At the start of the period of straining both systolic and diastolic pressure rose and the pulse pressure remained unchanged,

FIG. 2.—Distribution of systolic upstroke times and pulse pressures in resting arterial pulse tracings from 40 patients with aortic valve disease.

FIG. 3.—The relationship between systolic upstroke time and pulse pressure in resting arterial pulse tracings in 40 patients with aortic valve disease.

but after three to five pulses the systolic and diastolic pressures began to fall. The delay in the onset of the fall in pulse pressure was similar in aortic stenosis and in aortic incompetence: the fall in systolic pressure was greater than the fall in diastolic pressure so that the pulse pressure always fell. During this period the anacrotic notch, when present, initially remained at about the same height above the diastolic pressure, although towards the end of the straining period it usually fell (Fig. 4A). The fall in systolic pressure, occurring simultaneously, led to a reduction in the distance between the anacrotic notch and the systolic peak. Towards the end of the period of straining, when the falls in pulse pressure were maximal, the anacrotic notch disappeared. During the recovery phase patients in all three groups developed an overshoot, with a rise in pulse pressure, and as it rose the anacrotic notch reappeared and moved down the ascending limb of the pressure pulse curve.

In some resting tracings, two peaks were present. Their relative magnitude did not indicate whether the patient had stenosis, incompetence, or a combined lesion. During the straining period the second peak always became smaller, and usually formed a notch on the descending limb (Fig. 4B). Later it disappeared so that the pulse became single peaked, and during the overshoot this process was reversed. In three patients with aortic stenosis, one severe, the anacrotic notch moved towards the summit so that two equal peaks became evident: the second peak then became smaller as described above.

The Relationship between Changes in Pulse Pressure and in Systolic Upstroke Time. In those with aortic stenosis, the systolic upstroke time shortened when the pulse pressure fell during straining; in those with aortic incompetence it showed little consistent change. The changes in systolic upstroke time were similar when the changes in pulse pressure were due to atrial fibrillation.

The changes in systolic upstroke time for change in pulse pressure were measured. Two examples of the scatter of points so obtained are shown in Fig. 5. Regression lines of these changes were calculated for each patient, and the lines obtained in a group of seven patients with severe

FIG. 4.—An example of the alteration in the pulse form during the Valsalva manoeuvre in two patients, (A) with aortic stenosis and (B) with aortic stenosis and incompetence.

FIG. 5.—Examples of the scatter of points obtained by plotting pulse pressure against systolic upstroke time, (1) in a patient with severe aortic stenosis (coefficient of correlation $+0.9002$) and (2) in a patient with moderately severe aortic stenosis (coefficient of correlation $+0.971$). The regression formulae are shown, where S is systolic upstroke time and P the pulse pressure. The time unit of S is in 0.01 sec.

FIG. 6.—The regression lines of four patients with mild or moderately severe aortic stenosis (interrupted lines), and of seven patients with mixed aortic incompetence and stenosis (dark lines).

stenosis are shown in Fig. 7. In all the lines are steep, giving a large change in systolic upstroke time for change in pulse pressure. The lines obtained in a group of patients with aortic incompetence are contrasted in Fig. 7: they are very flat, being almost parallel to the pulse pressure axis, indicating that change of pulse pressure is associated with little or no change in systolic upstroke time in this group.

Four of the milder group with aortic stenosis, described above, were also studied in this way and the regression lines obtained are shown in Fig. 6. While not as steep as those found with severe aortic stenosis, they are not as flat as those found with aortic incompetence. Of the seven patients with mixed aortic stenosis and incompetence, four had flat regression lines resembling those found in subjects with aortic incompetence and three had lines resembling those of the patients with mild stenosis, being intermediate between those with severe stenosis and those with incompetence (Fig. 6).

In four patients, regression lines were obtained before and after aortic valvotomy (Fig. 8). In all four the slope of the regression line was much flatter after valvotomy than it had been before. In four other patients lines could be obtained only after operation: none were as steep as those obtained in the severe stenotic group. One line was flat; and it is of interest that severe aortic incompetence was present after the valvotomy in this patient: the other three were steeper, resembling those found with mild stenosis.

The Relationship between Change in Pulse Pressure and Rate of Systolic Upstroke. Variations in pulse form during the Valsalva manœuvre may lead to difficulty in measurement of the systolic upstroke time, particularly when a double summit is present. To overcome this we tried to determine whether the change in systolic upstroke time resulted solely from the change in pulse form

FIG. 7.—The regression lines of 7 patients with severe aortic stenosis, and 7 patients with aortic incompetence.

FIG. 8.—An example of the change in the regression line of a patient with severe aortic stenosis, following aortic valvotomy.

FIG. 9.—An example of the relationship between the *initial* rate of rise, and of the *mean* rate of rise of the pulse, and the pulse pressure in one patient with aortic stenosis and in one with aortic incompetence.

or partly also from the change in the rate of rise of the pulse wave. To do this the rate of rise in mm. Hg/sec. of the first 0.06 second of the pulse wave was compared with the mean rate from the start of the rise to the systolic peak, and the changes in these measurements that occurred with change in pulse pressure were also compared. The results indicate that when the pulse pressure changes, both the initial rate of rise and the mean rate of rise of the pulse wave alter in a similar manner. In aortic stenosis the rate of the pulse wave alters in a similar manner. In aortic stenosis the rate of rise alters much less for change in pulse pressure than it does in aortic incompetence. There is no doubt that the changes in pulse form, such as the disappearance of the second peak, are in part responsible for the reduction in systolic upstroke time with reduction of pulse pressure in aortic stenosis. There is equally no doubt, however, that the changes in pulse pressure also lead to changes in the speed of the upstroke of the pulse wave. The difference between patients with aortic stenosis and those with aortic incompetence, apply equally whether the initial rate of rise is measured or whether the total systolic upstroke is considered (Fig. 9).

DISCUSSION

The results of this work indicate that arterial pulse tracings taken under resting conditions, have a limited value in the assessment of aortic valve disease. The systolic upstroke time is almost always prolonged in aortic stenosis, but may not differ much in those with severe and in those with moderate or mild stenosis. Similarly, the pulse pressure in severe aortic stenosis is often below 50 mm., but the occurrence of pulse pressures of this order in milder stenosis and the occasional finding of greater pulse pressures in the severe group diminish the value of the pulse pressure in the assessment of the severity of the stenosis.

The relationship between systolic upstroke time and the pulse pressure in resting tracings often fails to help in assessment of the severity of aortic stenosis. When, however, the pulse pressure changes, either because of atrial fibrillation or during the Valsalva manœuvre, the relationship between the changes in pulse pressure and the changes in systolic upstroke time seems to be of value in determining whether the stenosis is severe or whether it is mild or associated with significant incompetence. In general, the greater the change in upstroke time for a given change in pulse pressure, the more severe is the stenosis likely to be. When this relationship is expressed as a regression line, the lines of those with severe stenosis are much steeper than the lines of the milder cases, and there is little overlap between the two groups. Furthermore, following aortic valvotomy the slope of the regression line is much flatter than before the operation, even when the resting upstroke time is little reduced.

Probably the fall of pulse pressure during the Valsalva manœuvre is due to the fall in stroke volume. In support of this is the fact that regression lines obtained in the same subject are almost identical, whether the variations of pulse pressure are due to atrial fibrillation or to the fall in pressure during the Valsalva manœuvre. If the variations in pulse pressure reflect changes in stroke volume, the linear relationship between change in systolic upstroke time and change in pulse pressure, and the apparent relationship between the slope of the regression line and the degree of aortic valve resistance suggest that the systolic upstroke time is a resultant of the stroke volume and valve resistance.

It must be emphasized, however, that although variations in pulse pressure in individual subjects probably reflect changes in stroke volume, differences in pulse pressures between different subjects cannot be taken to indicate corresponding differences in stroke volume; other factors such as aortic pressure-volume characteristics and the peripheral resistance also influence pulse pressure. These factors probably account for the lack of correlation between resting upstroke time, or resting pulse pressure, and the severity of the stenosis. They also affect the slope of the regression line in aortic stenosis, for the slope is steeper during the straining period than during the overshoot, probably because of the difference in peripheral resistance during the two periods. Another factor that must be taken into account is change of heart rate; it seems likely that alteration in rate would lead to variation in upstroke time by changing left ventricular stroke output.

The changes in pulse form that occur during the Valsalva manœuvre are of great interest. Goldberg *et al.* (1954) noted similar changes and ascribed them to change in pulse pressure. We believe that they are at least partly due to changes in peripheral vascular resistance, for pulses of identical magnitude recorded at different stages of the Valsalva manœuvre are often of very different form. Whatever their significance these variations suggest that the form of the pulse tracing in resting tracings may not be a reliable guide to the nature of the valve disease present, for the same subject may exhibit successively an anacrotic notch, a double summit pulse, and a smooth single peak. For these reasons no absolute value can be estimated for the slope of the regression line in severe aortic stenosis. Using the line obtained during the fall in pulse pressure, all the patients with severe stenosis had steep regression lines, giving a change in upstroke time of 0.10 sec. or more for a change of 50 mm. Hg in pulse pressure.

The delay in the fall in pulse pressure during the straining period, which was regarded by Gorlin and Case (1956) as characteristic of aortic stenosis, has not been evident in our patients; the delay in the onset of the fall was similar in those with mild or severe stenosis and in those with aortic incompetence. Our results differ also from those of Hamilton *et al.* (1936) in that all patients with aortic incompetence studied developed an overshoot after straining. We can offer no explanation for the difference between their results and ours, but it is of interest that Goldberg *et al.* (1954) noted that some with aortic incompetence developed overshoot responses after straining.

In spite of the theoretical complexities of the relationship between pulse pressure and upstroke time, the relationship between the changes in the two measurements seem useful as an index of severity of aortic stenosis. This information cannot be reliably deduced from resting pulse tracings, and even the measurement of the gradient across the aortic valve (Goldberg *et al.*, 1955) may be misleading in the presence of high or low left ventricular stroke volumes or in the presence of atrial fibrillation. We believe that the method of assessment reported here compares favourably in simplicity and accuracy with other methods at present available for the assessment of aortic valve function.

SUMMARY AND CONCLUSIONS

Although in aortic stenosis the systolic upstroke time is usually prolonged and the pulse pressure often low, neither measurement alone correlates well with the severity of stenosis.

The Valsalva manœuvre leads to variation in the pulse form. The shape of the pulse is an unreliable guide to the relative dominance of stenosis or incompetence.

Variations in pulse pressure during the Valsalva manœuvre or in atrial fibrillation and variations of upstroke time in the same pulses have a linear relationship. In severe stenosis the upstroke time changes more than in milder stenosis for a given change in pulse pressure. The relationship provides an index of severity of stenosis.

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